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INHERITANCE AND INTERACTION OF GENES FOR DISEASE RESISTANCE IN BEANS (PHASEOLUS VULGARIS L.)

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1. INTRODUCTION

TESTING OF BEANS for resistance has often given no results of practical importance, when the knowledge of inheritance was restricted to a single disease only. In

connection with physiologic specialization of parasites, a plant breeder has to overcome the consequences of interaction of genes for disease resistance. Moreover, in order not to lose genes of value he has to select consecutively for resistance to different diseases. Hence for testing multiple resistance a special selection scheme was developed in the Netherlands. The diseases concerned were anthracnose, halo blight, common bean mosaic, yellow bean mosaic, and stipple streak (14).

2. ANTHRACNOSE (COLLETOTRICHUM LINDEMUTHIANUM (SACC. ET MAGN.) BRIOSI ET CAV.)

In 1942 Andrus and Wade (2) studied the factorial interpretation of anthracnose resistance in beans. For the physiologic races beta and gamma they proposed a system of ten genes in three allelomorphic series, involving both duplicate and complementary genes for resistance, one dominant gene for susceptibility, and gene interactions at three points. For delta three independent pairs of genes would suffice as a simpler explanation, though the more complex system was not excluded. They could not study the inheritance of resistance to alpha, because all well-known resistant varieties proved susceptible to the alpha race they used.

Probably the physiologic forms studied by Andrus and Wade have to be understood as groups of races. Independently, Waterhouse (1955) (12) and Yerkes and Ortiz (1956) (13) described many new races. Nevertheless with few difficulties these could be classed into the four groups already mentioned. For this classification of races some American bean varieties were used as is shown in Table 1. Taking into account that differences in virulence of the races in each group may occur, there is some resemblance of beta and gamma races. Furthermore the narrow conformity of alpha and delta races is striking. One could suppose delta to be a more virulent alpha and likewise gamma a more virulent beta.

Sometimes morphological differences and similarities can be noticed too. On pepton agar our alpha and delta races mostly produced much black mycelium

TABLE 1

CLASSIFICATION OF PHYSIOLOGIC RACES OF COLLETOTRICHUM LINDEMUTHIANUM (SACC. ET MAGN.) BRIOSI ET CAV.

	Fungus races											
	alpha				delta		beta			gamma		
Bean varieties	1	2	3	4	5	6	7	8	9	10	11	12
Michelite, Robust	+	+	+	+	+	+	−	−	−	−	−	−
Perry Marrow	−	+	+	+	+	+	−	−	−	+	+	+
Red Kidney	−	−	+,−	+	+	+	+	+	−	+	+	−
Kievit	−		+,−			−	+			+,−		

1. alpha Barrus (1911)
2. II Yerkes & Ortiz (1956)
3. alpha mutant Hubbeling (1959)
4. alpha Andrus & Wade (1942)
5. delta Andrus & Wade (1942)
6. delta Hubbeling (1957)
7. beta Barrus (1911)
8. ACEFGH Waterhouse (1955)
9. I Yerkes & Ortiz (1956)
10. gamma Burkholder (1923)
11. B D Waterhouse (1955)
12. III Yerkes & Ortiz (1956)

− = resistant + = susceptible

with setae between which spores developed rather slowly and often scarcely. In contrast beta and gamma races normally showed such a rapid and abundant sporulation, that hardly any or no black mycelium and setae were formed. This is in agreement with a more general occurrence in the field of beta and gamma races than of races of alpha and delta, notwithstanding the fact that far more bean varieties appeared to be resistant to beta and gamma than to alpha and delta. Hence the selection under field conditions apparently was only possible in respect to beta and gamma races.

Some races of the fungus lost their virulence when they were maintained on artificial media for a long time. Characteristic in this respect was the production of sterile white or greyish woolly mycelium in the case of alpha and delta, whereas only with beta and gamma races sometimes intensely pink spores of a smaller size than normal were found. At temperatures over 25° C. these phenomena could often be found in parts of the cultures; below 20° C. they occurred only very seldom. A decrease of virulence could always be noticed when these types of degeneration manifested themselves. When delivering cultures to practical breeders in the past, these were not always kept under moderate temperature conditions. In such cases we could establish a decrease in virulence even surpassing the border from gamma to beta. On the other hand, under good conditions last year one race of alpha suddenly became so virulent that alpha and delta resistant varieties partly were attacked. These experiences proved that mutation or somatic segregation of genes for virulence of the fungus occurred commonly.

Therefore the question rose whether breeding for resistance to anthracnose would be possible at all. This became more probable when some Dutch plant breeders had already selected lines of beans, completely resistant to normal alpha, beta, gamma, and delta races. One breeder, having hundreds of such lines, tested them separately in 1959 using spore mixtures with and without the mutant alpha.

The result was that only one in about sixteen lines appeared to be completely resistant, whereas nearly all the rest segregated into slightly susceptible and resistant plants. This means that apparently two new genes for virulence of the parasite modified the activity of two genes for resistance of the host. A few years ago there were similar experiences, when some hundred lines still segregating resistant plants suddenly appeared to be susceptible. At that time a new and more virulent race of gamma was used in the spore mixtures. Nevertheless in both cases the same breeder could select a sufficient amount of completely resistant plants in crosses of beta and gamma resistant varieties with alpha and delta resistant ones, in respect to the new races.

In lines of the third and following generations we could count clearly defined mendelian ratios of surviving resistant or slightly susceptible plants on the one hand and very susceptible, rapidly dying, plants on the other. Independent inheritance of three, four, five, and six major genes were determined respectively, which prevented young seedlings from total necrosis. Moreover, often two modifying genes proved to govern the symptom expression of surviving partly resistant seedlings. In accordance with Andrus and Wade we could not confirm the existence of genes in allelomorphic series, but only with regard to more and less virulent races of the same group.

In the absence of modifying genes, probably a hypersensitivity reaction takes place in the outer cells of young stems with germinating spores of all races.

Often such completely resistant stems exhibit a limited necrosis of some epidermal cells and a development of a thin corky layer at the outside of the cortex. The percentage of these resistant plants was different depending on the parents used in bean crosses. In certain crosses some genes for resistance always seemed to be inactivated a little by interaction with dominant modifying genes.

Under poor conditions of light and temperature resistant plants sometimes gave rise to the development of definite brown lesions in the stems. In this case partly resistant plants showed deep, brown spots.

In order to avoid unexpected interactions of genes of host plants with genes of different races of the parasite, consecutive tests during at least four generations are necessary.

3. HALO BLIGHT. PSEUDOMONAS PHASEOLICOLA (BURKH.) DOWSON

For resistance to halo blight one or two genes with recessive inheritance are required, depending on the parents used, as Schuster (11) published in 1950. No linkage nor interaction of genes was observed. We could confirm the observations of Goss (6) in 1940, that symptom expression is highly governed by temperature conditions.

We isolated a strain of the bacterium however, which was unable to produce chlorosis in the leaves attacked under optimal conditions, but caused abundant greasy spots on the pods. Perhaps this strain lost its ability to form toxins, which induce chlorosis in the foliage of susceptible plants. In any case no other difference in reaction of resistant and susceptible bean varieties could be detected.

4. COMMON BEAN MOSAIC. PHASEOLUS VIRUS 1

Ali (1) reported in 1950 the presence of a recessive gene for resistance in some bean varieties and a dominant gene for hypersensitivity in other bean varieties, which sometimes develop black root but normally are resistant to common bean mosaic.

In 1957 Quantz (8) found, that at 30° C. detached leaves of hypersensitive varieties in petri dishes will exhibit black root symptoms.

In the field these symptoms occurred only under conditions of high temperature. They are mostly connected with very virulent strains of the virus.

In the absence of this dominant gene for hypersensitivity, Rudorf in 1958 (9) could demonstrate the existence of a second gene governing a recessive inheritance of resistance.

5. YELLOW BEAN MOSAIC. PHASEOLUS VIRUS 2

Rudorf in 1955 (10) could establish as many as three recessive genes for resistance in interspecific crosses of *Phaseolus vulgaris* and *Phaseolus coccineus*. Baggett and Frazier in 1957 (3) could determine two or three major recessive genes for resistance with modifiers in a variety of *Phaseolus vulgaris*. We observed that hybrids of highly tolerant very susceptible parents showed intermediate symptoms. Buishand (5) in 1955 concluded that perhaps a dominant gene prevents top necrosis. Different strains of the virus must be taken into account.

6. STIPPLE STREAK. NICOTIANA VIRUS 11

About the inheritance of resistance to stipple streak we only know that two or more recessive genes are necessary. We expect that different strains of the virus will be present in the field. Up to now only tolerance was found in *Phaseolus vulgaris*.

7. MULTIPLE RESISTANCE

Adding all desired genes for resistance to these five diseases we come to a number of at least sixteen. The impossibility of finding them together in one plant of the second generation of crosses between two varieties, which mutually possess resistance to five diseases, is clear. Combining only eight recessive genes in a segregating population actually means looking for one plant resistant to halo blight and three virus diseases against 65,535 susceptible plants. Selecting at the same time for resistance to no more than two of these diseases enlarges the chance to one desired plant among 256 plants.

Only a special scheme for selection and mutual recombination of resistant plants enables the plant breeder to increase gradually the number of genes desired. The following example may explain this.

Bean seedlings were tested in the first segregating populations for resistance to all races of anthracnose. The resistant plants were then inoculated with virus of common and yellow bean mosaic. Thus the reactions of these plants could be observed. From plants showing virus susceptibility, the seeds were harvested too, because they might still contain genes for resistance to other diseases. In further progenies plants with an increasing number of genes for virus resistance were selected after testing for anthracnose resistance. Resistance to halo blight and stipple streak was secured by testing leaves on old plants with the aim of not losing genes. In order to incorporate the multiple resistance and other desired characters, crosses of F_5 or F_6 lines were made, which showed already homozygotic resistance to anthracnose, common mosaic and halo blight, but which were different in resistance in respect to yellow mosaic and stipple streak. In this way only testing of the segregating populations of such crosses for resistance to the last two diseases is necessary.

Finally it can be concluded, that the knowledge of the inheritance of genes for resistance in beans enabled the breeders to test and select more efficiently, avoiding the risks of interactions. Only the pure plant pathologist will perhaps be satisfied when the parasites by their specialization are modifying the limits of genes for resistance.

REFERENCES

1. ALI, M.A. Phytopath. 40(1950): 69–70.
2. ANDRUS, C. F. and WADE, B. L. Tech. Bull. U.S. Dept. Agric. Wash. 810 (1942), 29 pp.
3. BAGGETT, and FRAZIER, W. A. Proc. Amer. Soc. Hort. Sc. 70(1957): 325–33.
4. BARRUS, M. F. *Phytopath.* 1(1911): 190–5.
5. BUISHAND, TJ. Proefst. Groentet. Alkmaar 1(1955), 48 pp.
6. GOSS, R. W. Phytopath. 30(1940): 258–64.
7. HUBBELING, N. Euphytica 6(1957): 111–41.
8. QUANTZ, L. Nachrbl. deutsch Pflschutzd. 9(1957): 71–4.
9. RUDORF, W. Phytop. Zeitschr. 31(1958): 371–81.
10. —— Rep. 14th Int. Hort. Congr. 1955: 446–51.

11. SCHUSTER, M. L. Phytopath. 40(1950): 604–12.
12. WATERHOUSE, W. L. Proc. Linn. Soc. N. S. W. 80(1955): 71–83.
13. YERKES, JR, W. D. and ORTIZ, M. T. Phytopath. 46(1956): 564–7.
14. ZAUMEYER, W. J. and REX THOMAS, H. A. A monographic study of bean diseases and methods for their control. Tech. Bull. U.S. Dept. of Agr. Wash. 868 (1957), 255 pp.